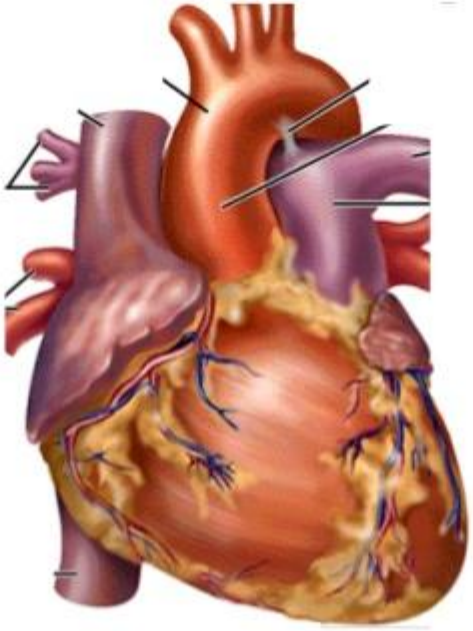
PHYSIOLOG OF HEART



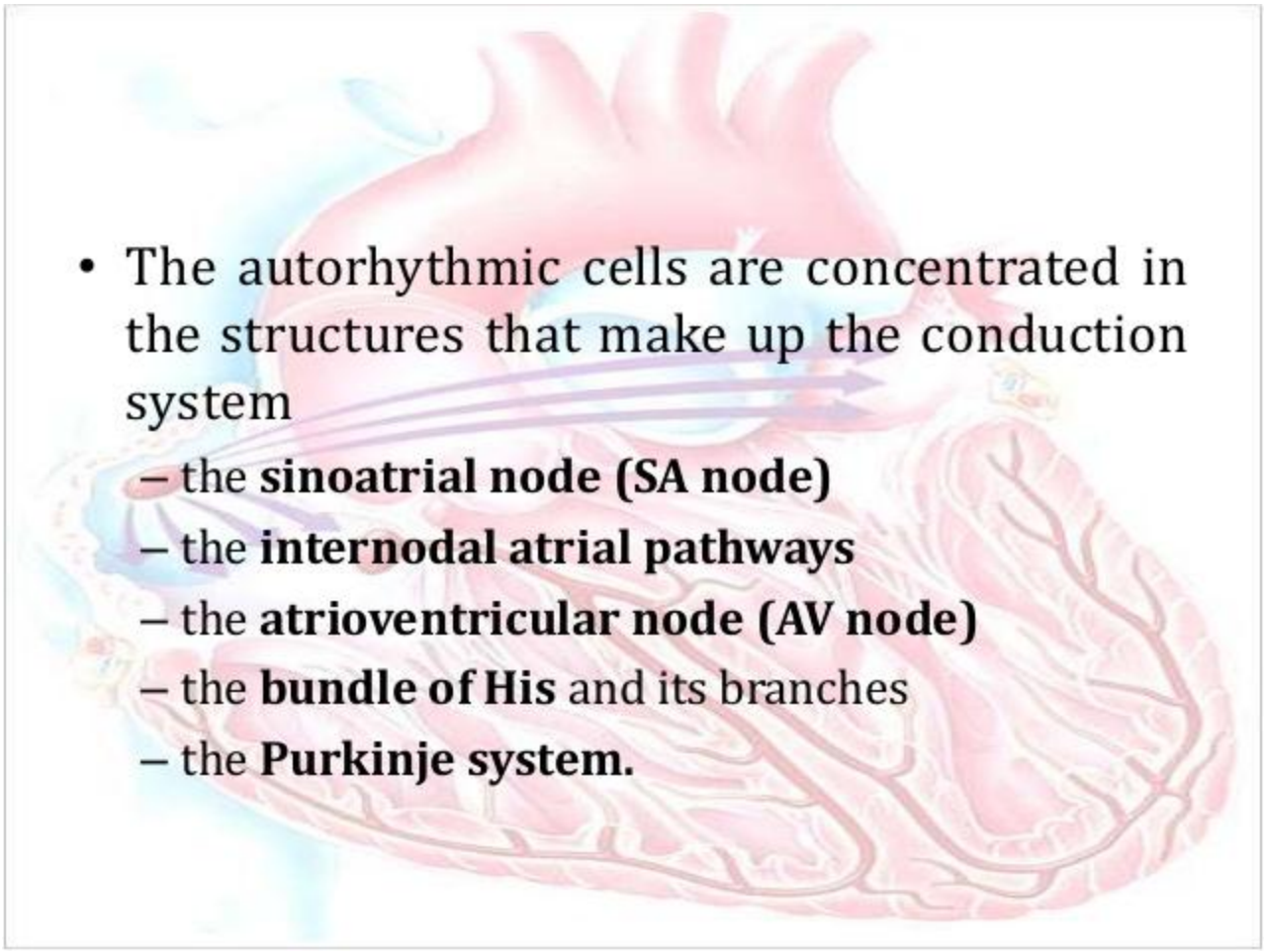
DR/AMAT

Electrical Activity of the Heart

- The heartbeat originates in a specialized **cardiac conduction system.**
- The heart beats normally in an orderly sequence: Contraction of the atria **(atrial systole)** is followed by contraction of the ventricles **(ventricular systole),** and during **diastole** all four chambers are relaxed.

CONDUCTION SYSTEM OF THE HEART

- Action potentials (electrical impulses) in the heart originate in specialized cardiac muscle cells, called **autorhythmic cells**.
- These cells are self-excitable, able to generate an action potential without external stimulation by nerve cells.
- The autorhythmic cells serve as a pacemaker to initiate the cardiac cycle (pumping cycle of the heart) and provide a conduction system to coordinate the contraction of muscle cells throughout the heart.



- The autorhythmic cells are concentrated in the structures that make up the conduction system
  - the **sinoatrial node (SA node)**
  - the **internodal atrial pathways**
  - the **atrioventricular node (AV node)**
  - the **bundle of His** and its branches
  - the **Purkinje system.**

Sino atrial node

- It is a part of the wall of right atrium close to the opening of superior venacava. It generates impulses approximately at the rate of 72 times/min. SA node is called the pacemaker because it depolarizes at a faster rate than any other group of cells in the heart, and imposes that faster rate on the heart as a whole.

Atrio ventricular node

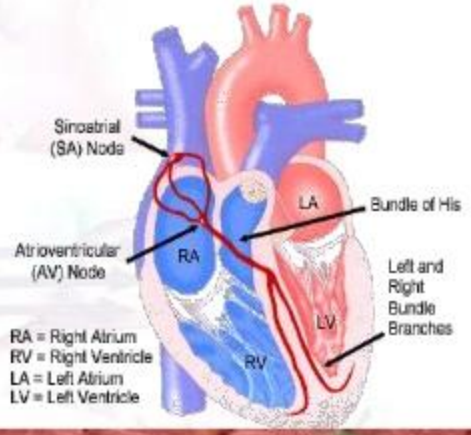
- It is a part of the wall of right atrium close to the atrioventricular septum and near to the tricuspid valve. It generates impulses approximately at the rate of 60 times/min.

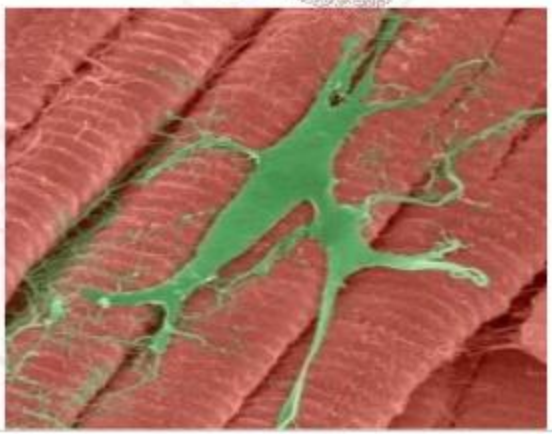
Bundle of His

- It is a thick band of muscle fibers starting from A.V Node. It runs along with intra ventricular septum. It divides into right and left bundle branch. It generates impulses approximately at the rate of 40 times/min.

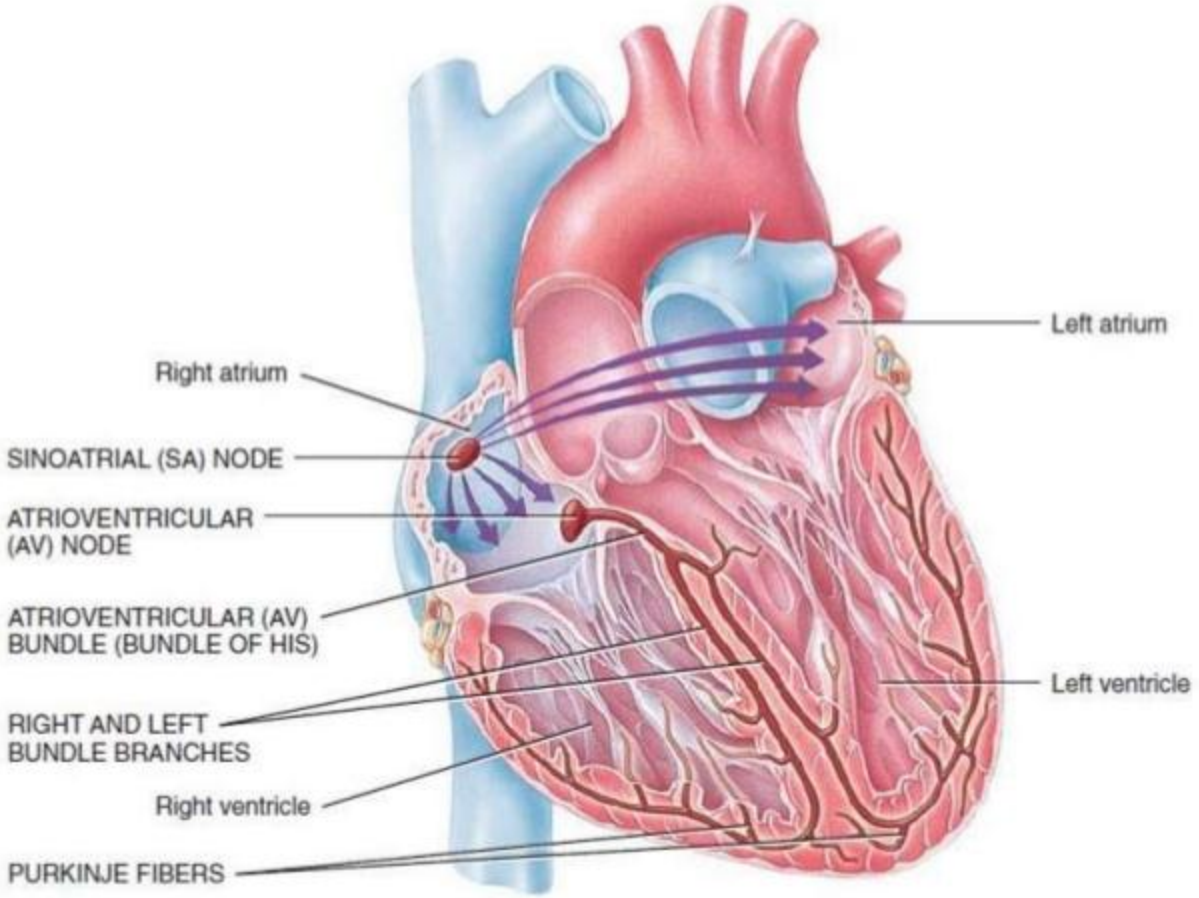
Purkinje Fibers

- These fibers arise from the branches of bundle of His. These fibers pierce into the ventricular myocardium.

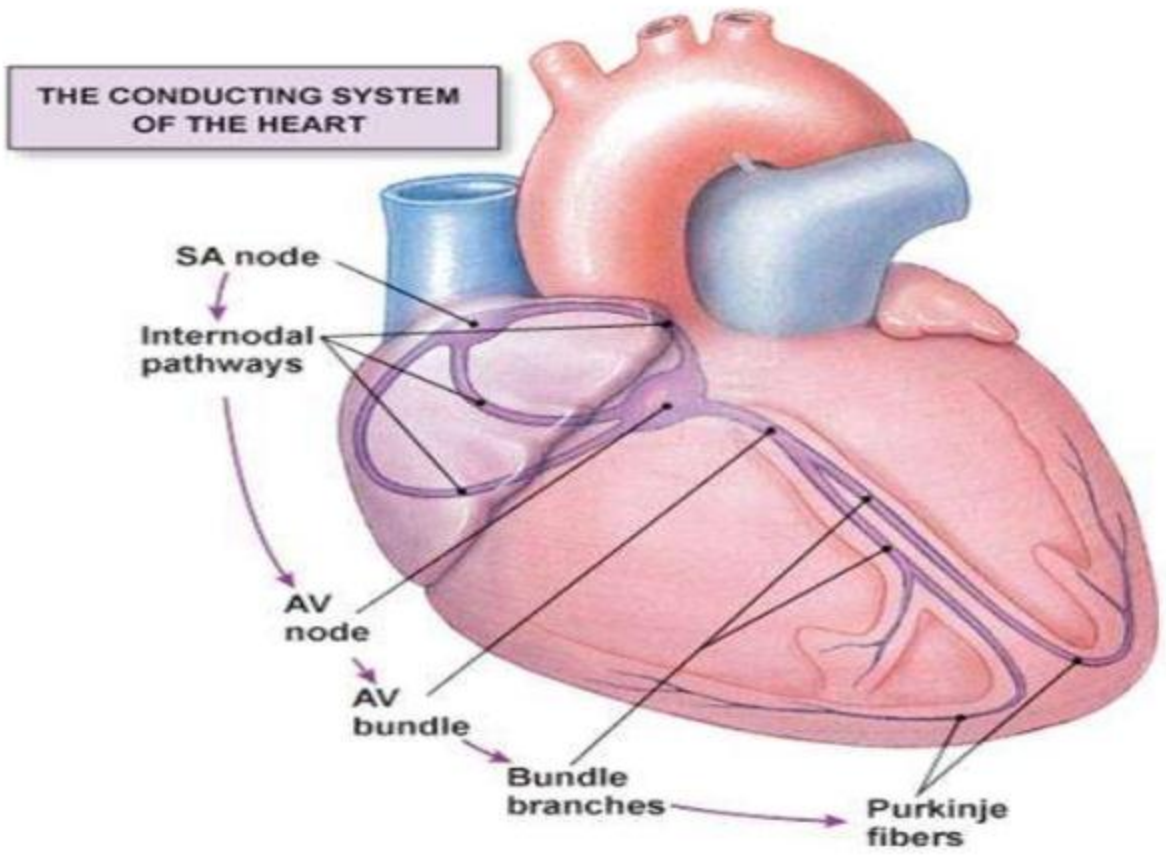




- Cardiac excitation normally begins in the **sinoatrial (SA) node, located in the right atrial wall just inferior and lateral to** the opening of the superior vena cava.
- The spontaneous depolarization is a **pacemaker potential.** *(The **pacemaker potential** is a slow, positive increase in voltage across the cell's membrane)*
- **When the pacemaker** potential reaches threshold, it triggers an action potential. Each action potential from the SA node propagates both atria via gap junctions in the intercalated discs of atrial muscle fibers and contract both atria at the same time.


Left atrium
Right atrium
SINOATRIAL (SA) NODE
ATRIOVENTRICULAR (AV) NODE
ATRIOVENTRICULAR (AV) BUNDLE (BUNDLE OF HIS)
Left ventricle
RIGHT AND LEFT BUNDLE BRANCHES
Right ventricle
PURKINJE FIBERS

- By conducting along atrial muscle fibers, the action potential reaches the **atrioventricular (AV) node, located in the interatrial** septum, just anterior to the opening of the coronary sinus.
- From the AV node, the action potential enters the **atrioventricular (AV) bundle (also known as the bundle of His**). This bundle is the only site where action potentials can conduct from the atria to the ventricles.


THE CONDUCTING SYSTEM OF THE HEART
SA node
Internodal pathways
AV node
AV bundle
Bundle branches
Purkinje fibers

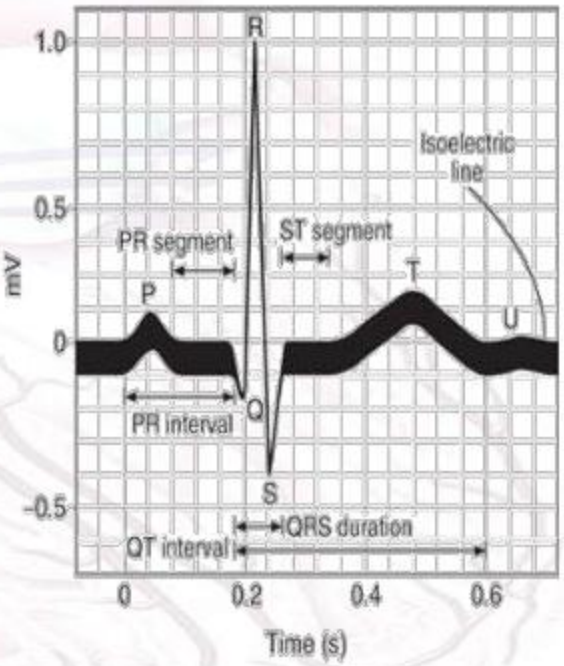
- After propagating along the AV bundle, the action potential enters both the **right and left bundle branches.**
- Finally, the large-diameter **Purkinje fibers** rapidly conduct the action potential beginning at the apex of the heart upward to the remainder of the ventricular myocardium. Then the ventricles contract, pushing the blood upward toward the semilunar valves.

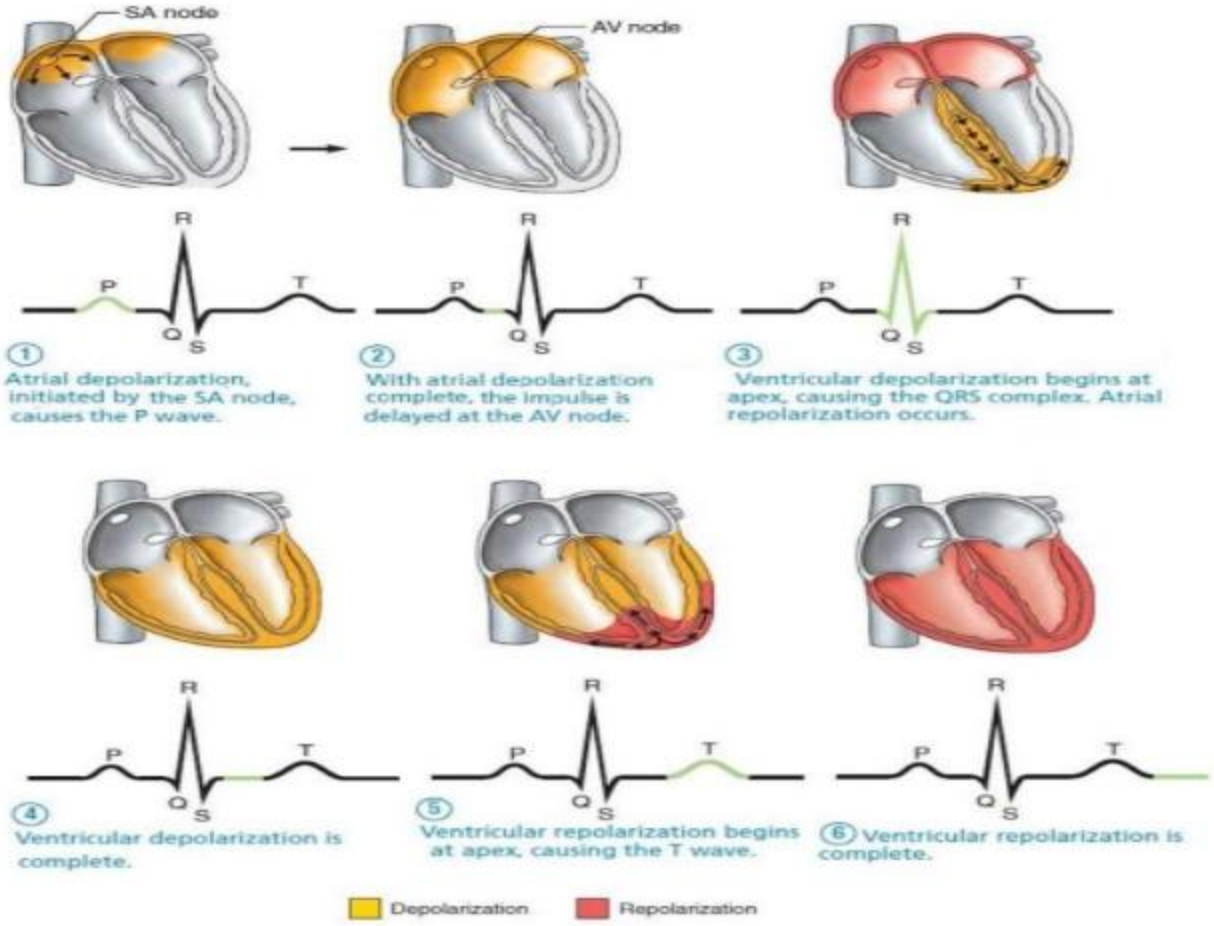
Conduction Speeds in Cardiac Tissue	
Tissue	**Conduction Rate (m/s)**
SA node	0.05
Atrial pathways	0.1
AV node	0.05
Bundle of His	0.1
Purkinje system	0.4
Ventricular muscle	0.1

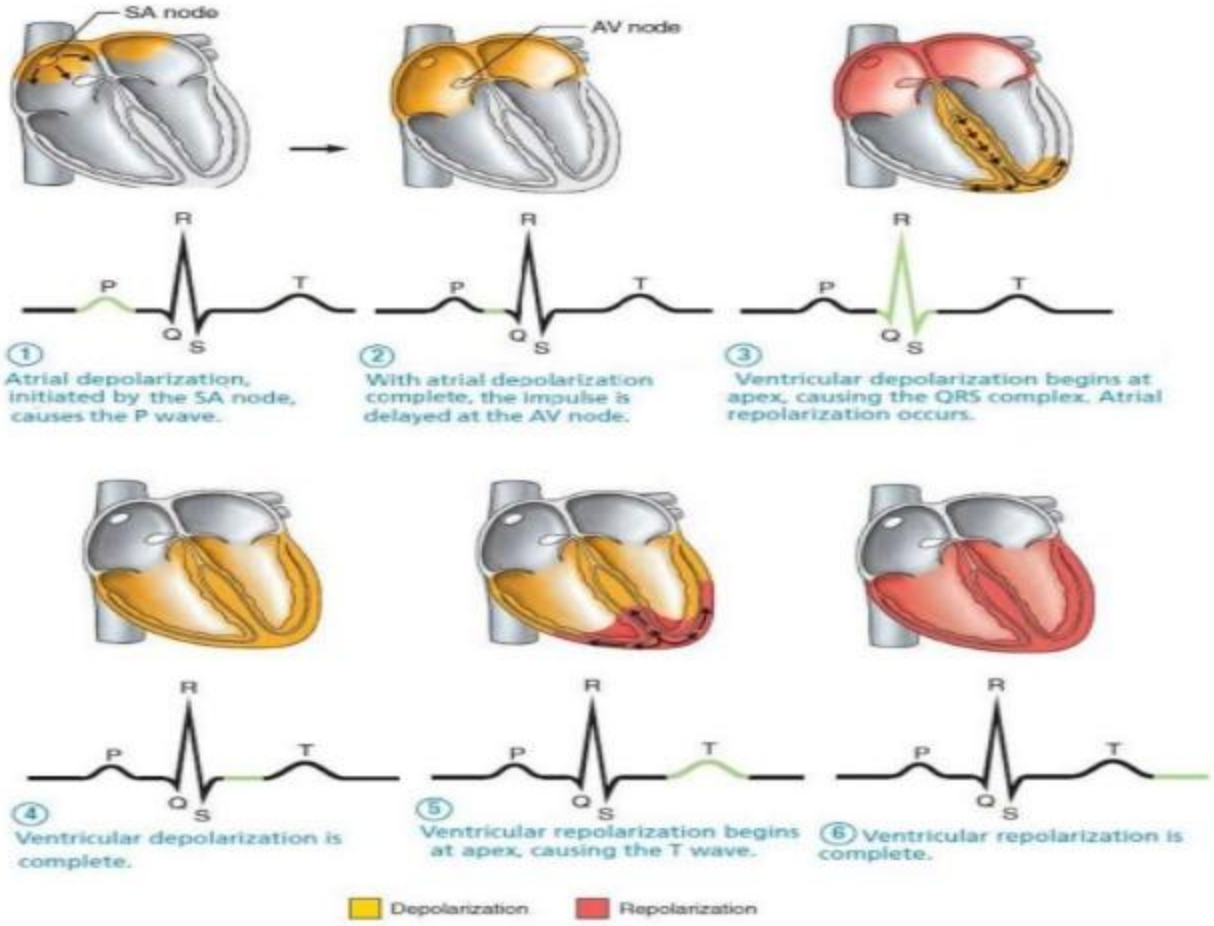
- **SA node** is called the **pacemaker of the heart**. If for any reason the SA node stops beating, the **AV node**, which has the next fastest rate of depolarization, would become the heart's pacemaker. If the AV node failed, the **bundle of His** would take over. If it failed, the **Perkinje fibers** would start the heartbeat, and if they failed as well, a group of cells somewhere else in the heart would start pulsing. If failed, eventually it can no longer sustain life.

The Electrocardiogram

- The body fluids are good conductors, fluctuations in potential that represent the algebraic sum of the action potentials of myocardial fibers can be recorded extracellularly.
- The record of these potential fluctuations during the cardiac cycle is the **electrocardiogram (ECG).**




SA node
AV node
R
P
T
Q
S
1
Atrial depolarization, initiated by the SA node, causes the P wave.
2
With atrial depolarization complete, the impulse is delayed at the AV node.
3
Ventricular depolarization begins at apex, causing the QRS complex. Atrial repolarization occurs.
4
Ventricular depolarization is complete.
5
Ventricular repolarization begins at apex, causing the T wave.
6
Ventricular repolarization is complete.
Depolarization
Repolarization


SA node
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Atrial depolarization, initiated by the SA node, causes the P wave.
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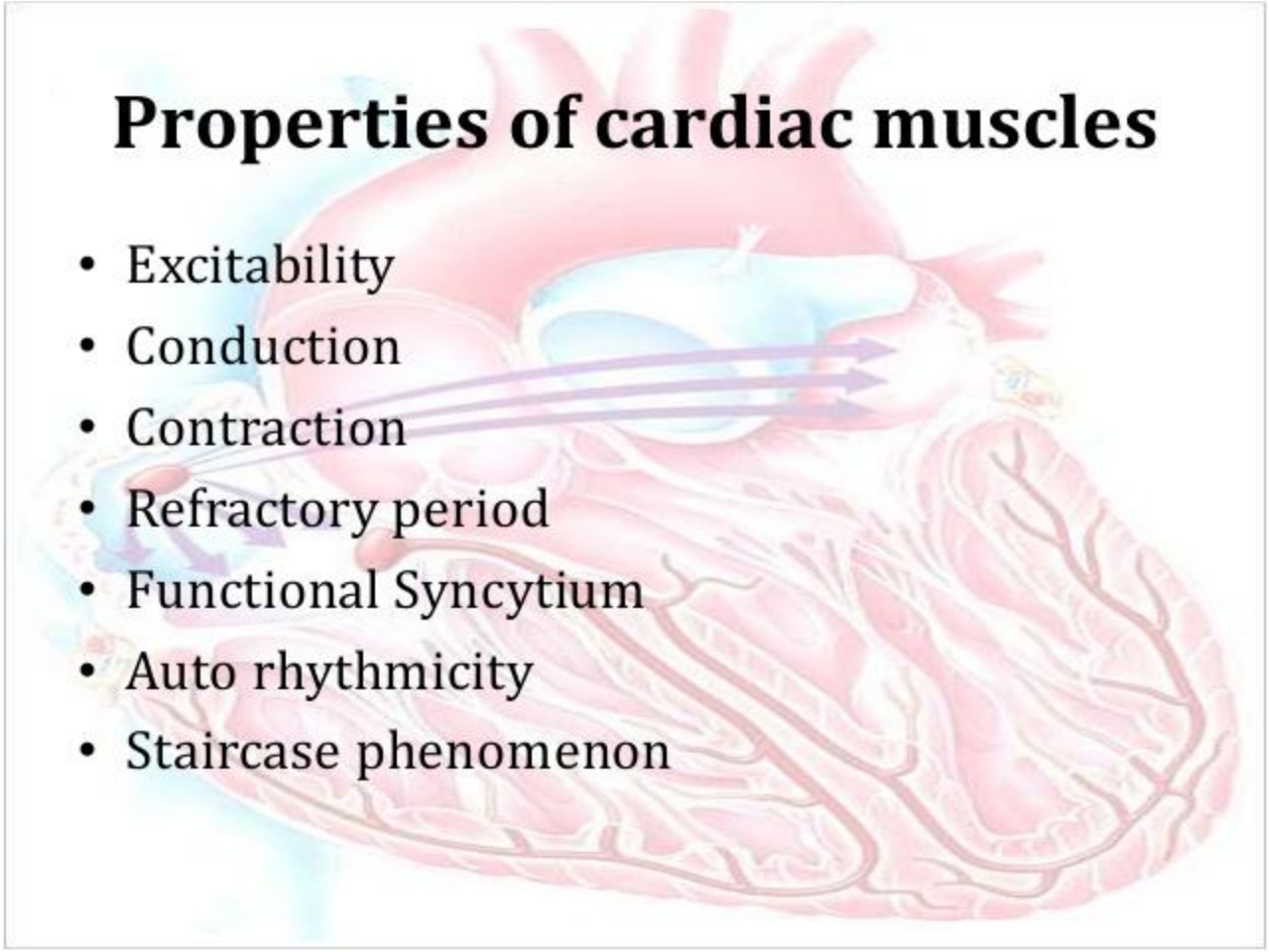
Ventricular depolarization begins at apex, causing the QRS complex. Atrial repolarization occurs.
4
Ventricular depolarization is complete.
5
Ventricular repolarization begins at apex, causing the T wave.
6
Ventricular repolarization is complete.
Depolarization
Repolarization

Properties of cardiac muscles

- Excitability
- Conduction
- Contraction
- Refractory period
- Functional Syncytium
- Auto rhythmicity
- Staircase phenomenon



Excitation

- It is an electrical event. Calcium ion are responsible for this event

Conduction

- The action potential is propagated all along the length of the muscle fiber this phenomenon is known as conduction.

Contraction

- It is the shortening of muscle fibres.

Refractory period

- It is the period during which the 2nd stimulus cannot generate a fresh action potential. It is divided in to two
  - Absolute refractory period (0.25 sec)
  - Relative refractory period (0.05 sec) it may generate an action potential.

Functional Syncytium

- Cardiac muscles act as single unit this phenomenon is called Functional Syncytium.

Auto rhythmicity

- Cardiac muscle can generate their own impulse, this property of heart is called the auto rhythmicity

Staircase phenomenon

- When a series of stimuli of the same intensity are sent into the muscle after a calm period, the first few contractions of the series show a successive increase in amplitude.

Cardiac Cycle

- Cardiac cycle is defined as sequence of cyclical changes taking place in the heart from one beat to the next.
- A cardiac cycle consists of systole and diastole of the atria plus systole and diastole of the ventricles.
- A cardiac cycle duration is 0.8 sec.

Changes during Cardiac cycle

1. Mechanical changes: contraction and relaxation of atria and ventricles.
2. Electrical changes
3. Volume change inside the heart
4. Pressure change inside the chambers
5. Opening and closing of valves
6. Heart sounds

- In each cardiac cycle, the atria and ventricles alternately contract and relax, forcing blood from areas of higher pressure to areas of lower pressure. As a chamber of the heart contracts, blood pressure within it increases.

Atrial Systole

- Depolarization of the SA node causes atrial depolarization which results atrial systole.
- During atrial systole, both the atria contracts at the same time, where as the ventricles are relaxed.
- The contraction of atrial muscles narrows the venacaval orifices and pulmonary vein orifices.
- And exert a pressure on the blood atria, which forces blood to move into ventricles through the open tricuspid and mitral valves.
- Atrial systole which lasts about 0.1 sec. The end of atrial systole is also the end of ventricular diastole (relaxation).
- Each ventricle contains about 130 mL at the end of its relaxation period (diastole). This blood volume is called the end-diastolic volume (EDV).

Ventricular Systole

- When the ventricles are contracting the atria are relaxed (atrial diastole).
- Ventricular depolarization causes ventricular systole. When ventricular systole begins, pressure rises inside the ventricles and pushes blood up against the atrioventricular (AV) valves, forcing them shut. For about 0.05 seconds, both the semilunar and AV valves are closed. This is the period of **isovolumetric contraction.**

- When left ventricular pressure exceeds aortic pressure (80 mmHg) and right ventricular pressure exceeds pressure in the pulmonary artery (20 mmHg), both semilunar valves open.
- At this point, ejection of blood from the ventricles begins.
- The left ventricle ejects about 70 mL of blood into the aorta and the right ventricle ejects the same volume of blood into the pulmonary trunk. The volume remaining in each ventricle at the end of systole, about 60 mL, is the **end-systolic volume (ESV).**
- Ventricular systole lasts about 0.3 sec.
- **Stroke volume (SV)** is the amount of blood ejected by the left ventricle in one contraction.
- **SV = EDV - ESV (130-60=70 ml)**

Relaxation Period (diastole)

- During the relaxation period, which lasts about 0.4 sec, the atria and the ventricles are relaxed. Blood flows into the heart throughout diastole, filling the atria and ventricles.
- Once the atrial muscles are relaxed venacaval orifices and pulmonary veins open and atria is filled blood.
- Once the ventricular muscle is fully contracted, the ventricular pressures drop rapidly. And blood in the aorta and pulmonary trunk begins to flow backward toward the regions of lower pressure in the ventricles.

- As the ventricles continue to relax, the pressure falls quickly. When ventricular pressure drops below atrial pressure, the AV valves open, and ventricular filling begins. Blood that has been flowing into and building up in the atria during ventricular systole then rushes rapidly into the ventricles. At the end of the relaxation period, the ventricles are about three-quarters full. The P wave appears in the ECG, signaling the start of another cardiac cycle.

Cardiac Output

- Cardiac output (CO) is the volume of blood ejected from the heart in each minute.
- Cardiac output is expressed in litres per minute (L/min).
- Cardiac output is the product of heart rate (HR) and stroke volume (SV).
- **CO= HR x SV**
- In a resting, supine man, it averages about 5 L/min (72 beats/min x 70 mL).

During exercise, venous return increases and stretches the ventricular myocardium, which in response contracts more forcefully, and results in increase stroke volume.

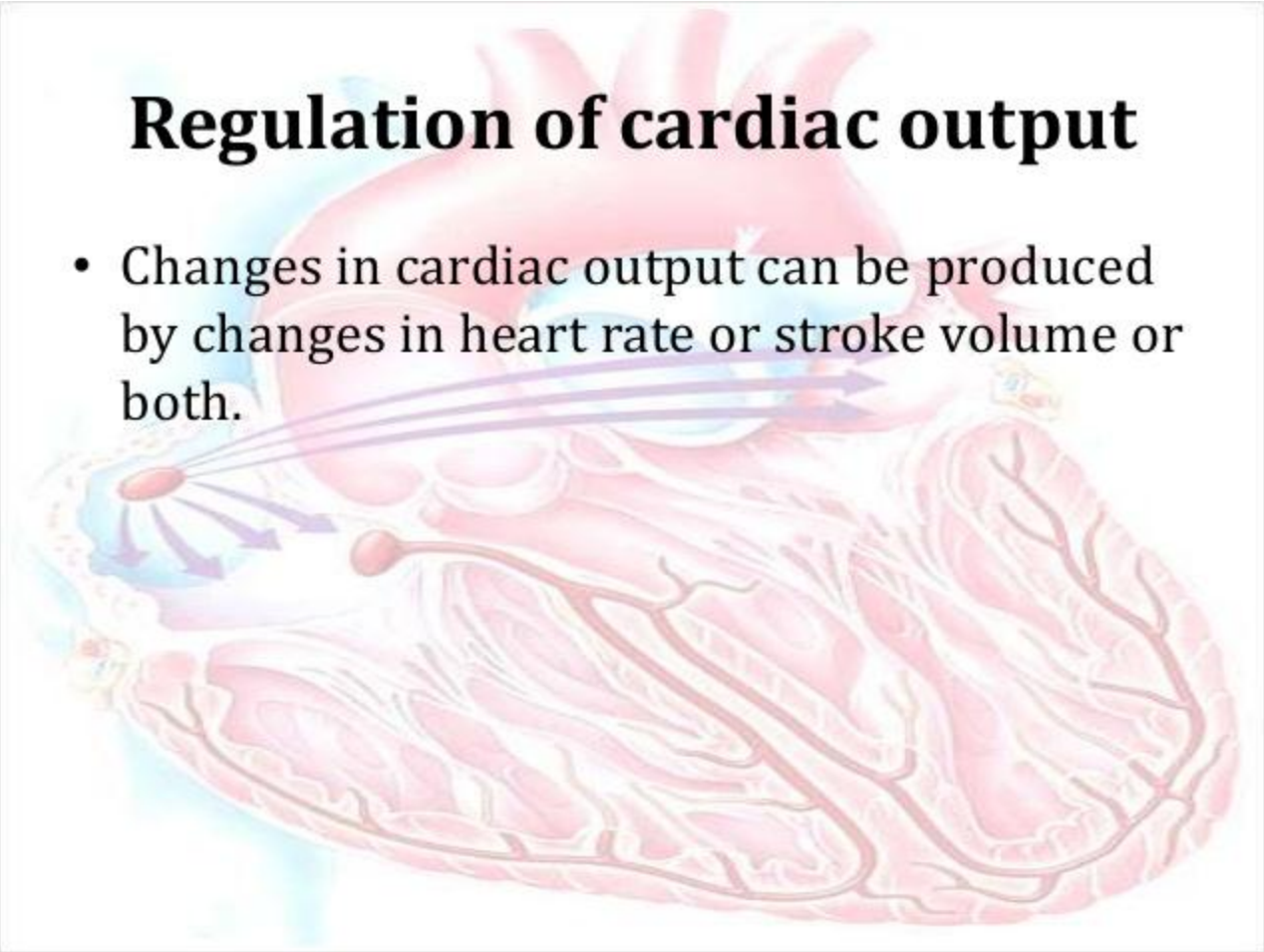
More blood is pumped with each beat, and at the same time, the heart rate increases, causing cardiac output to increase to as much as four times the resting level, and even more for athletes.

Factors affecting cardiac output

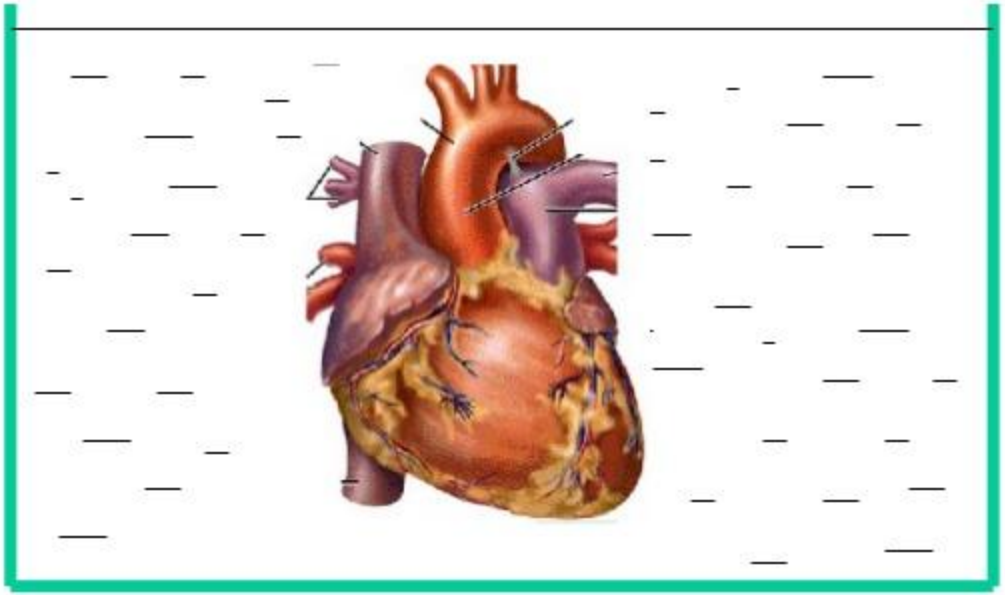
- Factors increases cardiac output
  - Anxiety and excitement
  - Eating
  - Exercise
  - High environmental temperature
  - Pregnancy
- Factors decreases cardiac output
  - Sitting or standing from lying position
  - Rapid arrhythmias
  - Heart disease

Regulation of cardiac output

- Changes in cardiac output can be produced by changes in heart rate or stroke volume or both.



REGULATION OF THE HEART FUNCTION



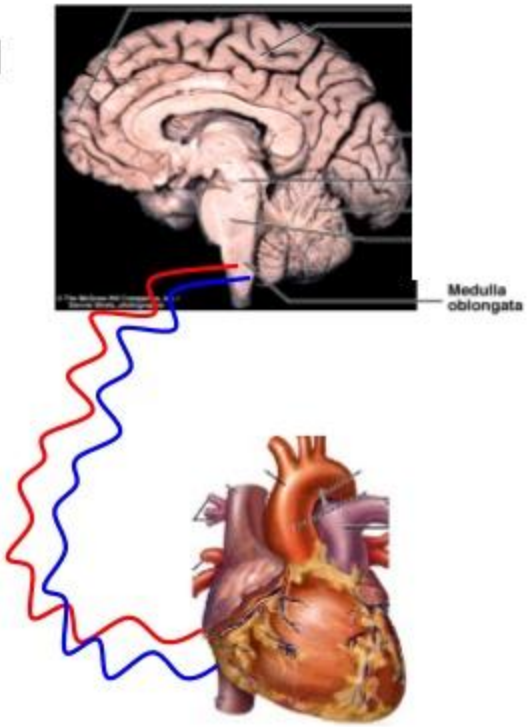
Regulation of the Cardiac Function

1) Nervous control
   - Sympathetic control
   - Parasympathetic control
   - Higher centers
   - Reflexes
2) Hormonal Control
3) Autoregulation
4) Other factors

Regulation of the Cardiac Function

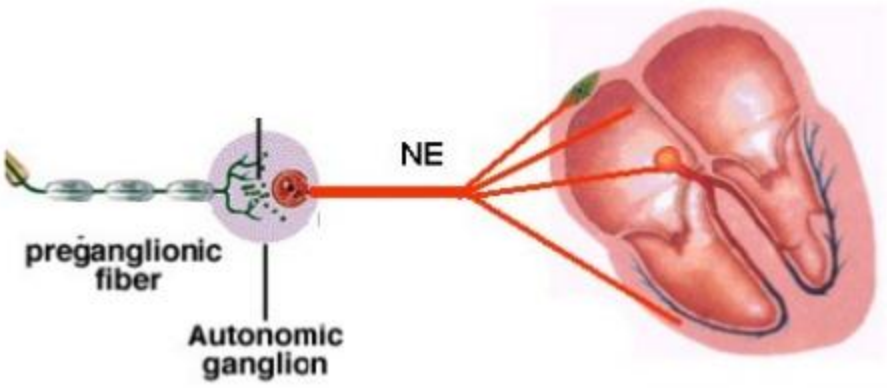
1) Nervous control

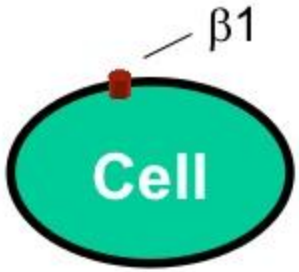
- Sympathetic control
- Parasympathetic control



Sympathetic Nervous System

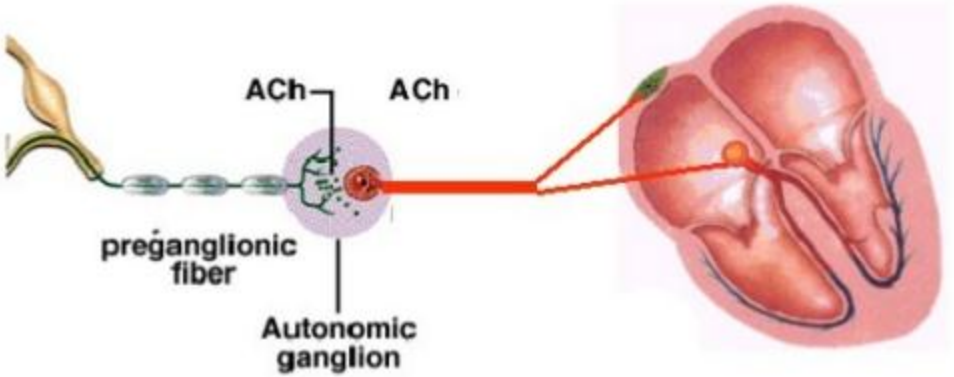
- controls all components of the heart.
- release ***Norepinephrine (NE).***
- increases heart rate (positive chronotropic) and contractility (positive inotropic).

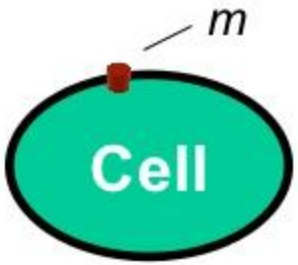




Parasympathetic Nervous System (PNS)

- controls SA node and AV node.
- releases ***Acetylcholine (Ach).***
- decreases heart rate (negative chronotropic).
- prolongs delay at AV node.
- has little effect on contractility.

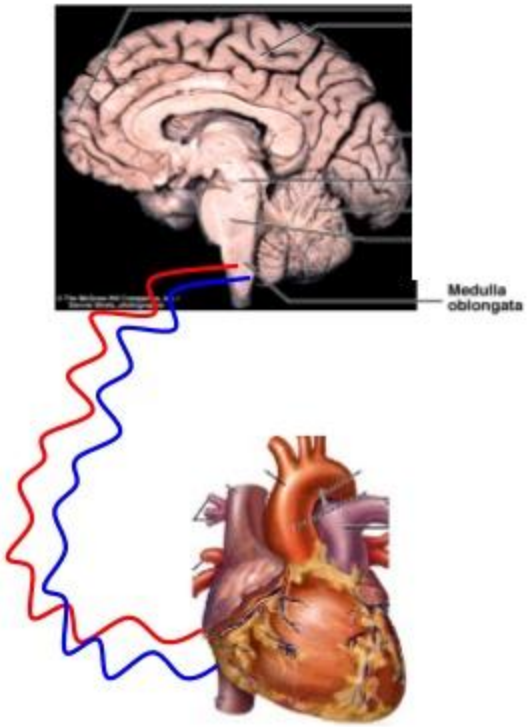




Higher Centers of Autonomic Nervous System

- **Medulla Oblongata**

- **Hypothalamus, Thalamus, Cerebral cortex**



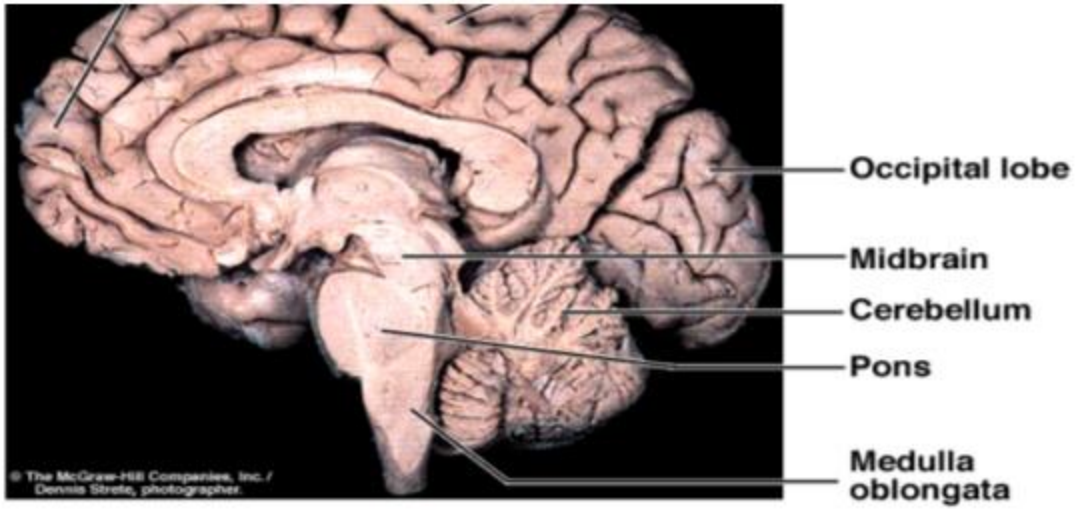
Centers in Medulla Oblongata

Sympathetic center:

distinct accelerator and augmentor

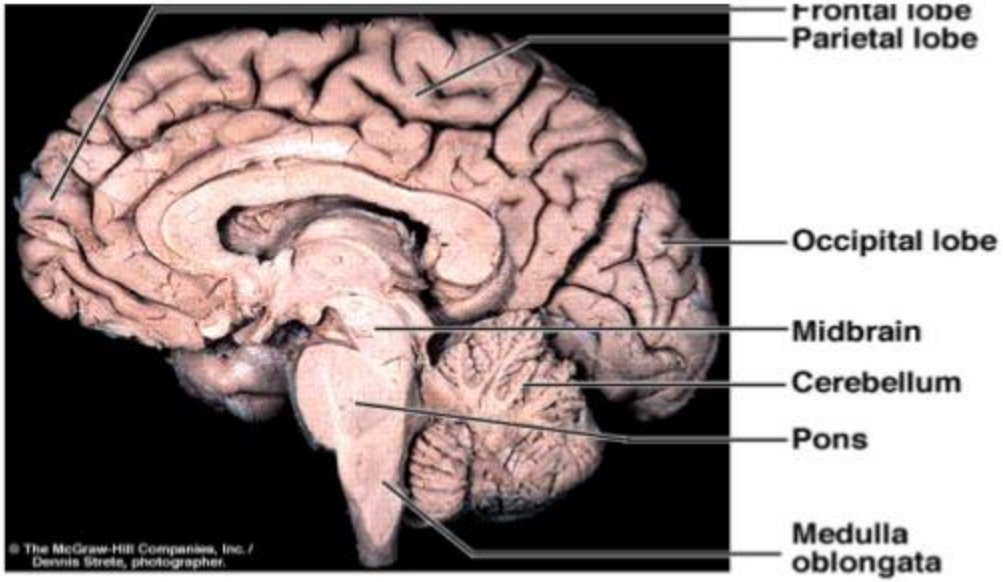
Parasympathetic center:

Nucleus vagus and nucleus ambiguus

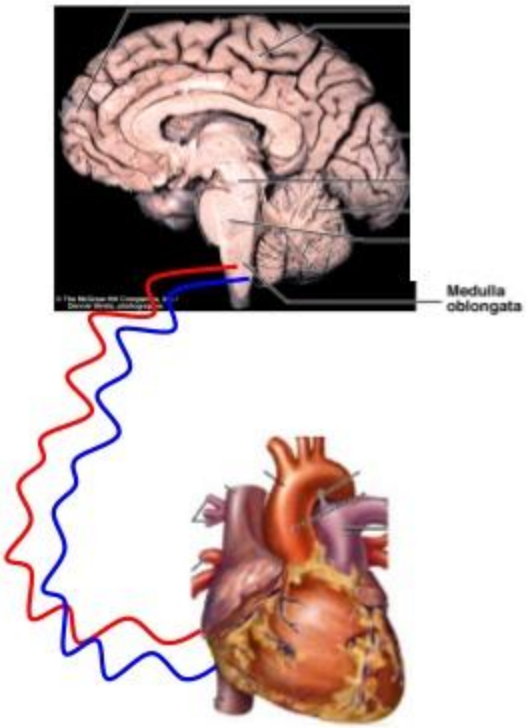


Hypothalamus, Thalamus, Cerebral cortex

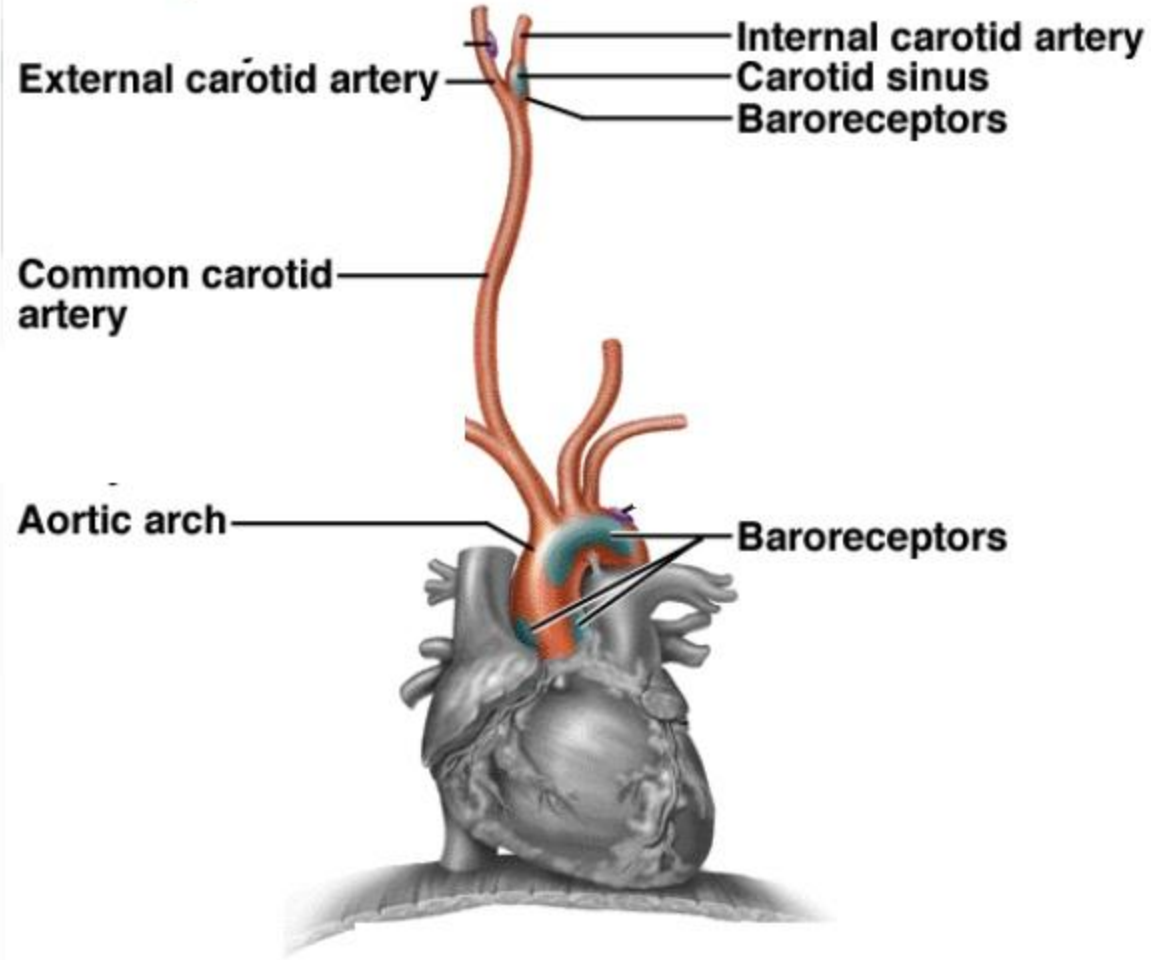
Involved in the cardiac response to environmental ***temperature*** changes, ***exercise***, or during ***excitement***, ***anxiety***, and other ***emotional*** states



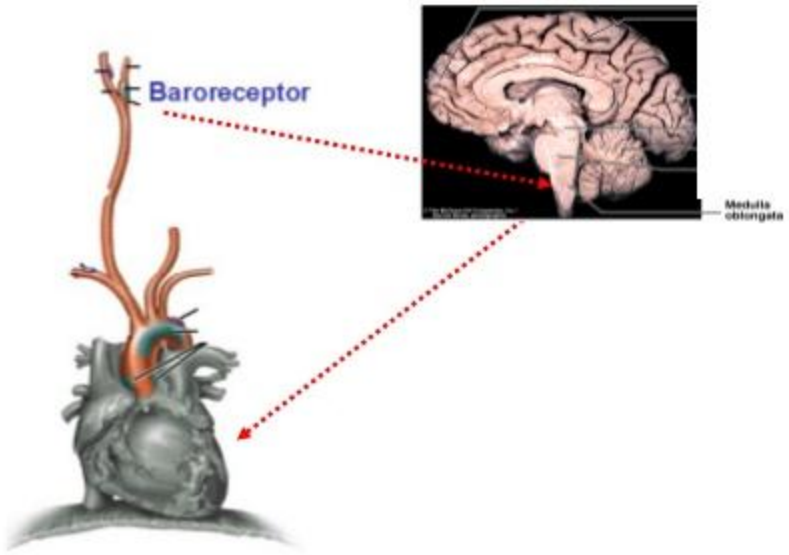
Neural Control via Reflexes



Baroreceptors

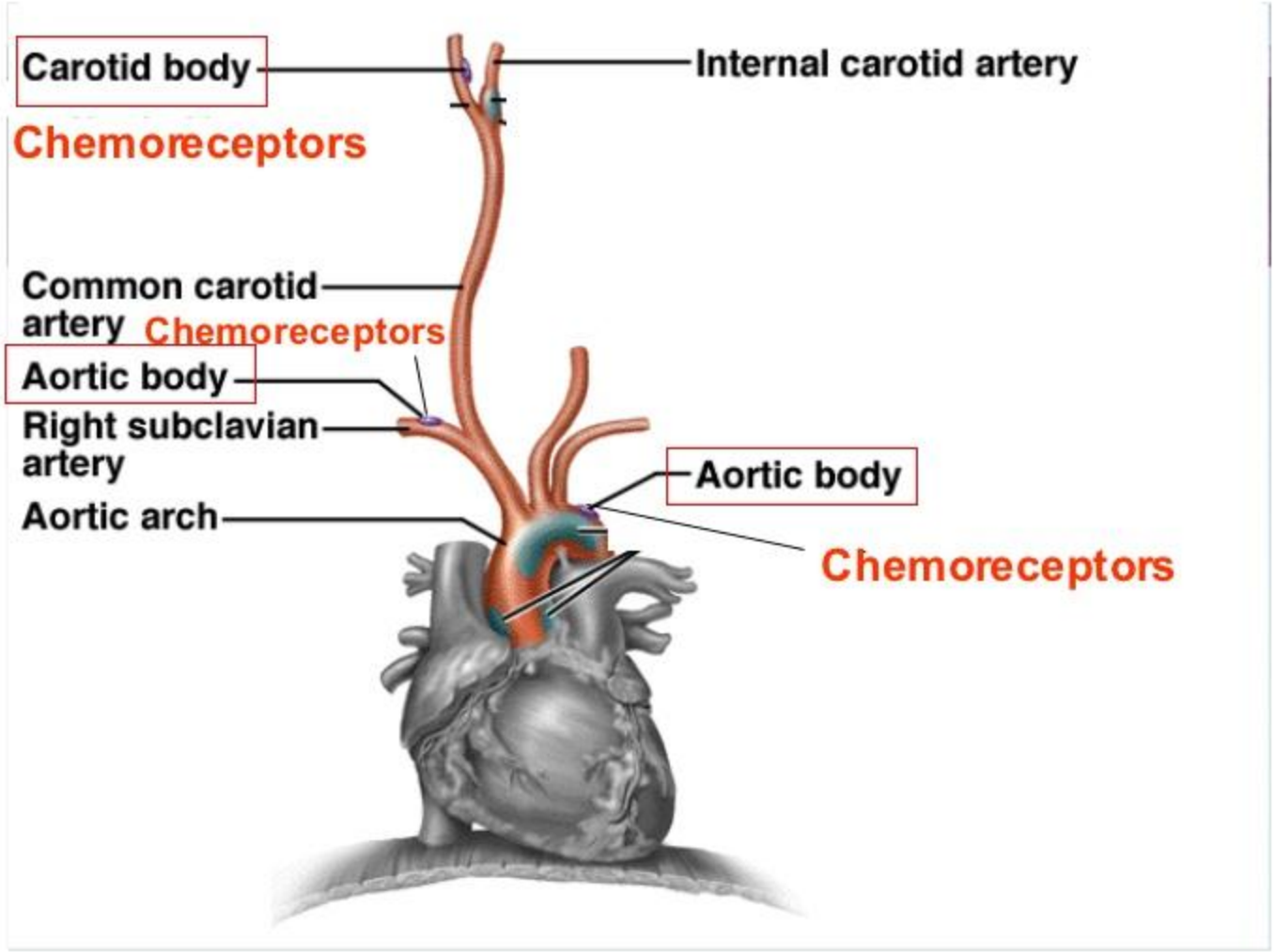


1) Baroreceptor Reflex

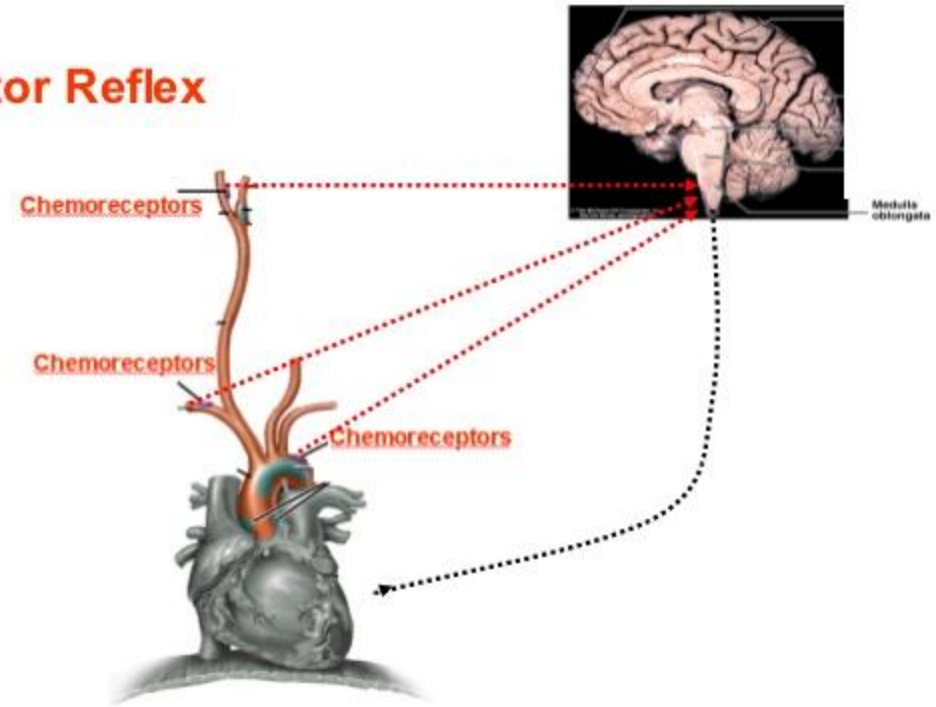


- stimulated by increase in arterial pressure (stretch)
- Effect: negative chronotropic and inotropic
- regulate the heart when BP increases or drops
- involved in short term regulation of BP

2) Chemoreceptor Reflex

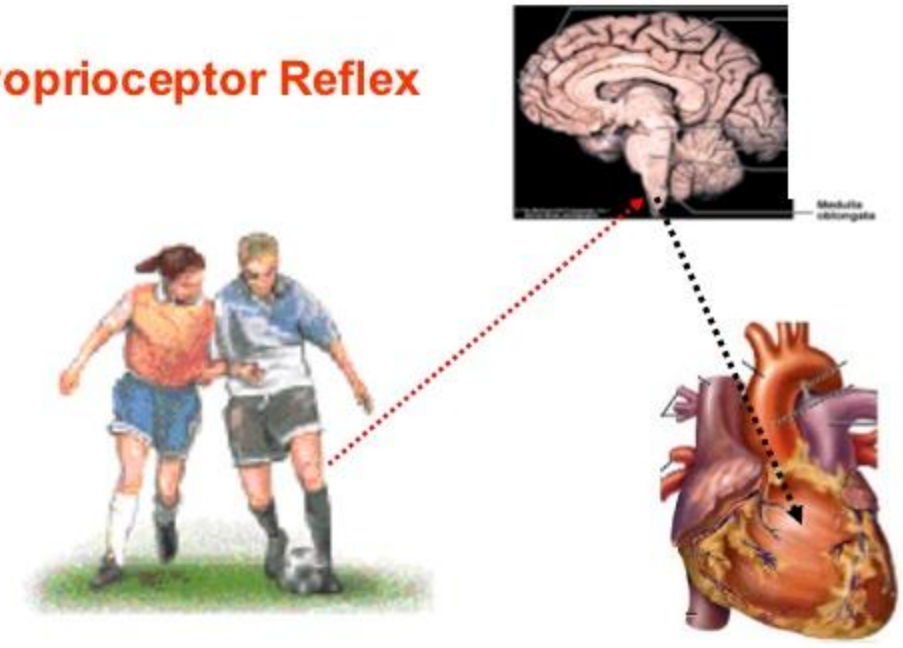

Carotid body
Internal carotid artery
Chemoreceptors
Common carotid artery
Chemoreceptors
Aortic body
Right subclavian artery
Aortic arch
Aortic body
Chemoreceptors

2) Chemoreceptor Reflex



- stimulated by ⇓***oxygen***, ⇓***pH***, or ⇑CO_2
- overall effect: positive choronotropic and inotropic.
- less important in regulating cardiac function

3) Proprioceptor Reflex

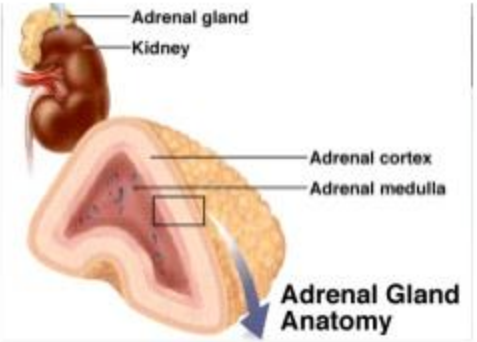


- Stimulated by muscle and joint movement
- Effects: increase heart rate during exercise

Regulation by Hormones

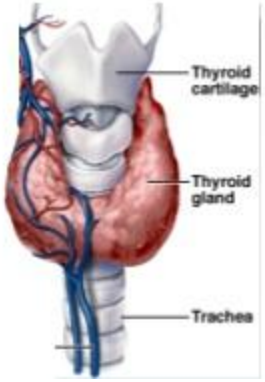
Epinephrine

- released from adrenal gland.
- increases heart rate and contractility.



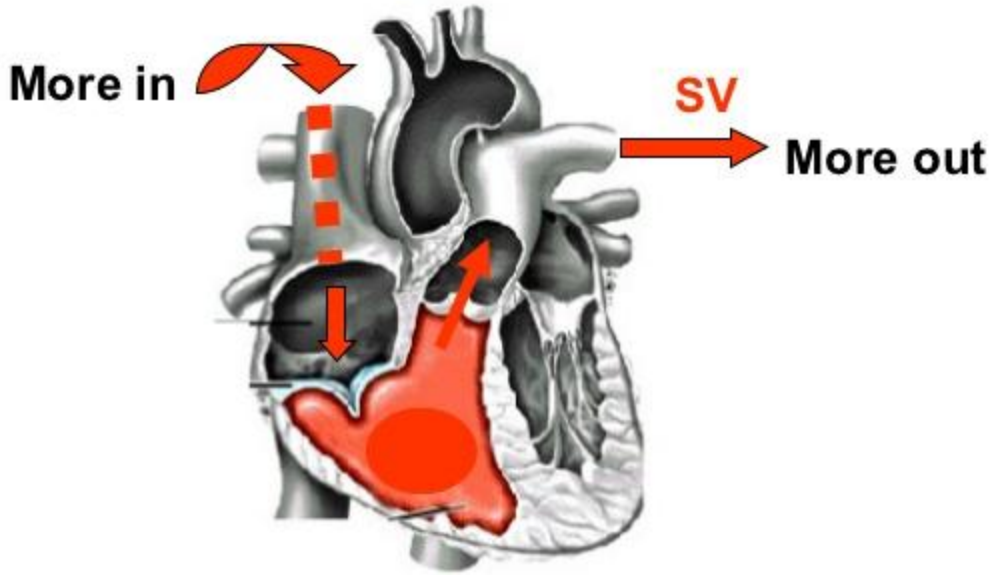
Thyroxin

- released from thyroid gland.
- increases heart rate.



Autoregulation of the Heart

Stroke volume is autoregulated by ventricular filling (Frank-Starling law).



4) Other Factors

- Blood level of ionic calcium, sodium, and potassium

Hypercalcemia (high plasma Ca^{++}):

positive inotropic

Hypernatremia (high plasma Na^{+}):

negative chronotropic

Hyperkalemia (high plasma K^{+}):

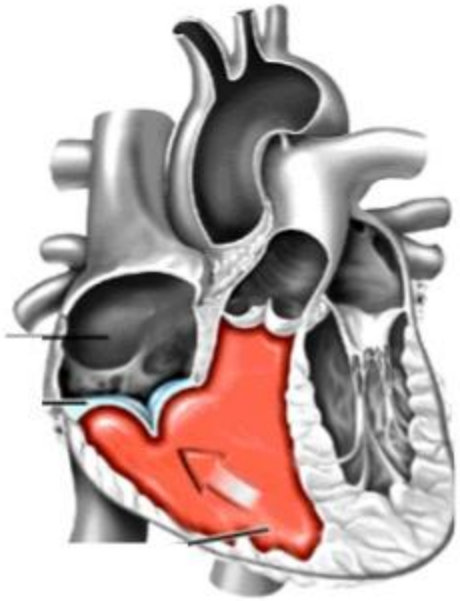
negative chronotropic

used in lethal injection

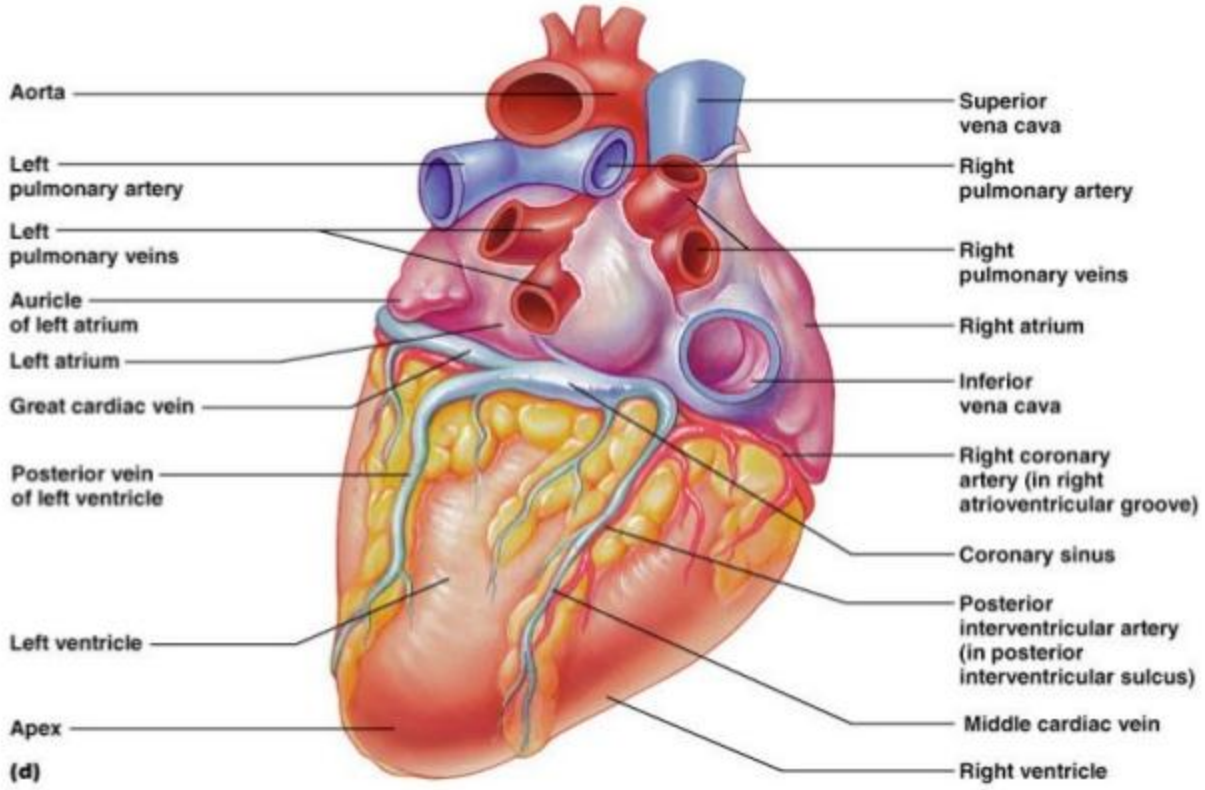
- Age, gender, exercise, and body temperature

Blood Supply to Cardiac Muscles

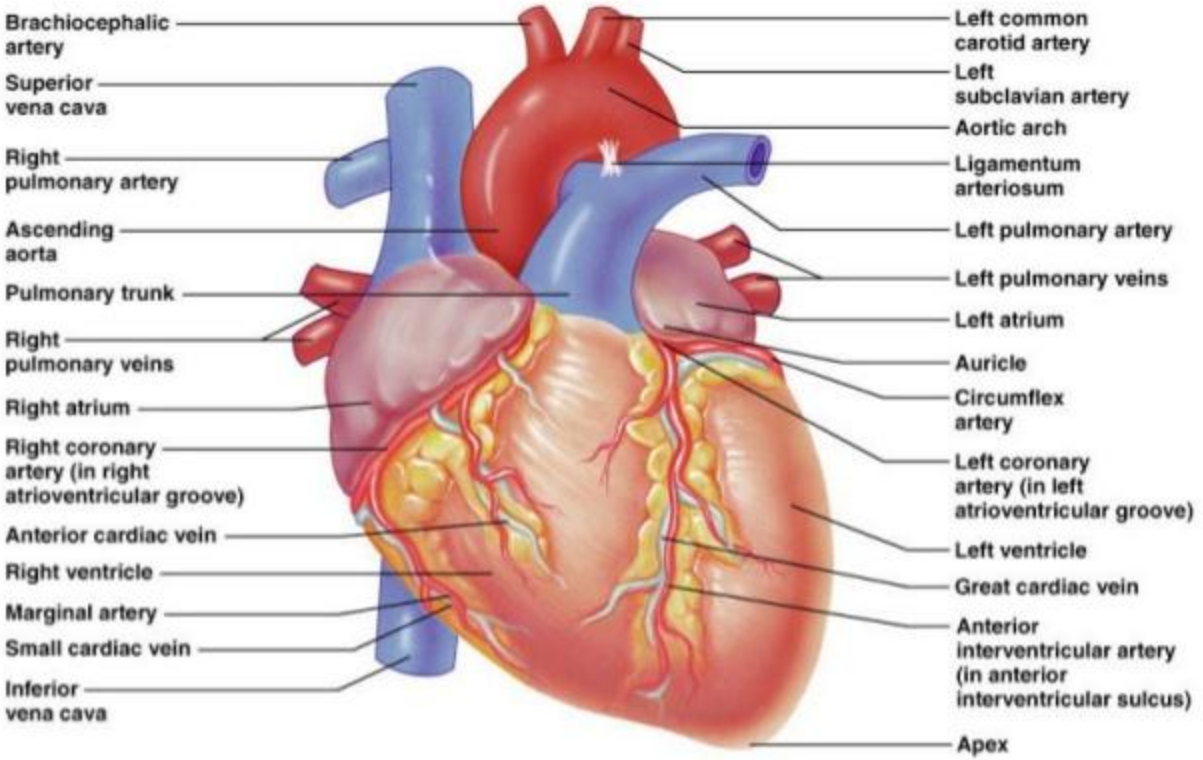
Can cardiac muscles get nutrients from the blood in heart chambers?



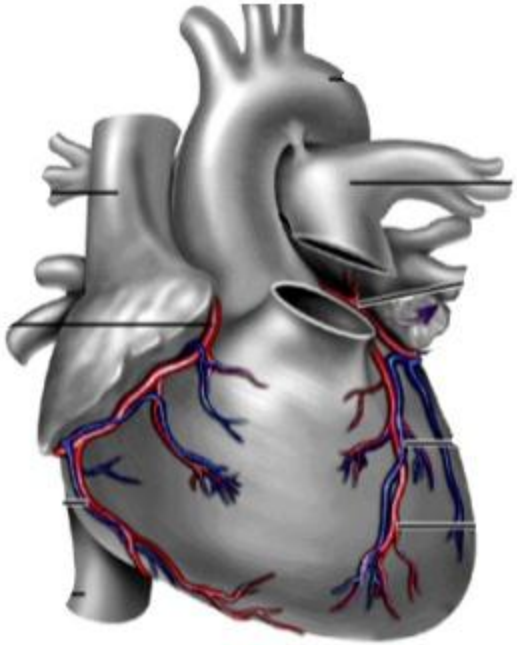
External Heart: Posterior View



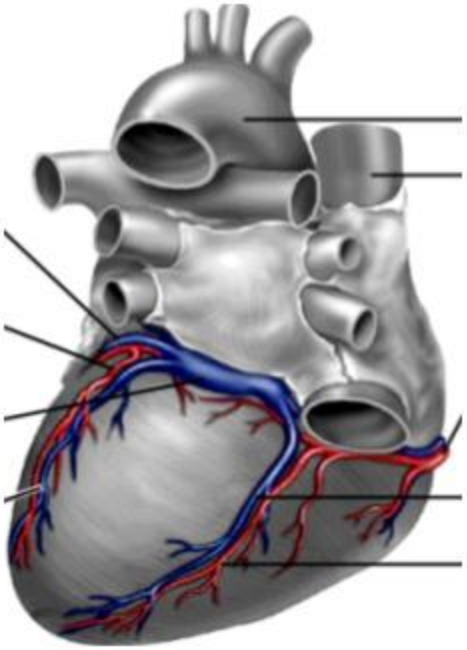
External Heart: Anterior View



The cardiac muscles get nutrients from coronary circulation.

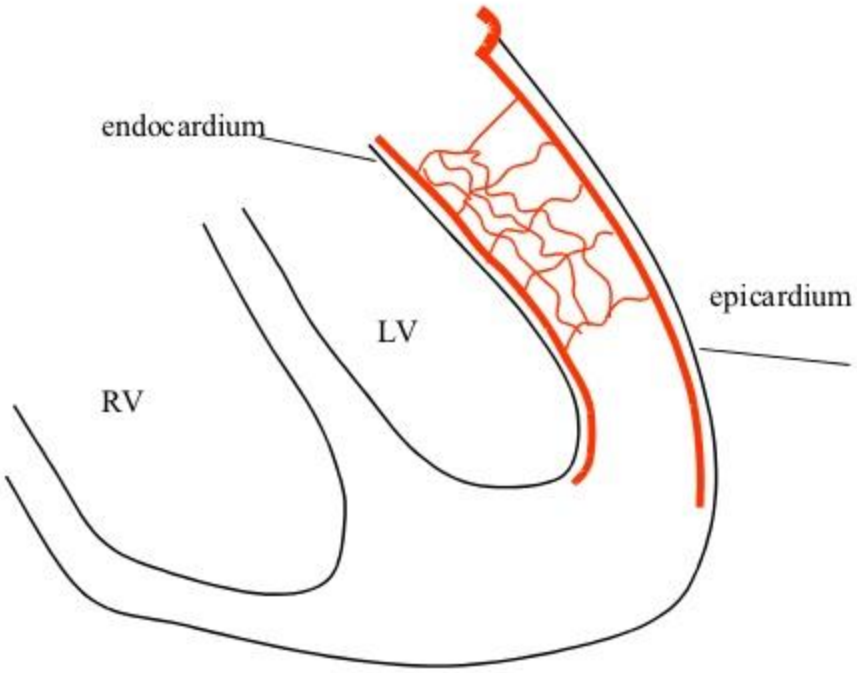


Anterior view

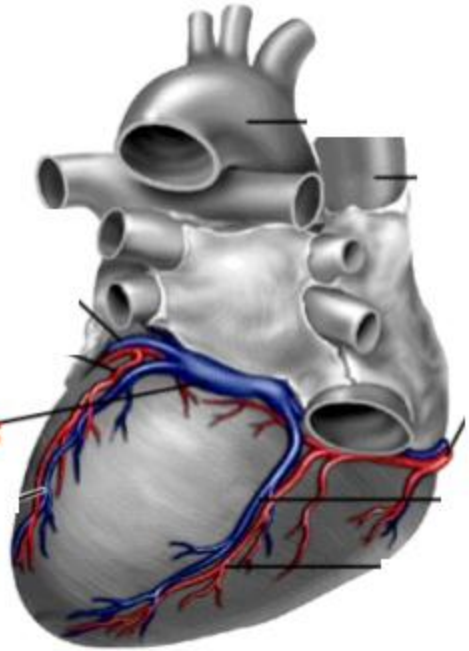


Posterior view

Coronary arterial anastomosis



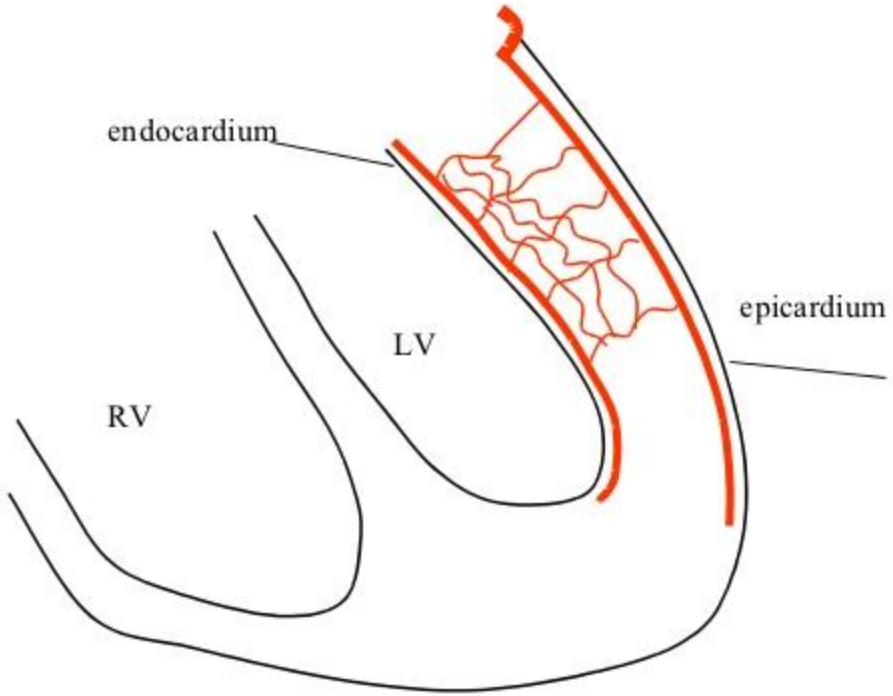
Coronary venous blood is emptied into the right atrium through cardiac veins and coronary sinus.



coronary sinus

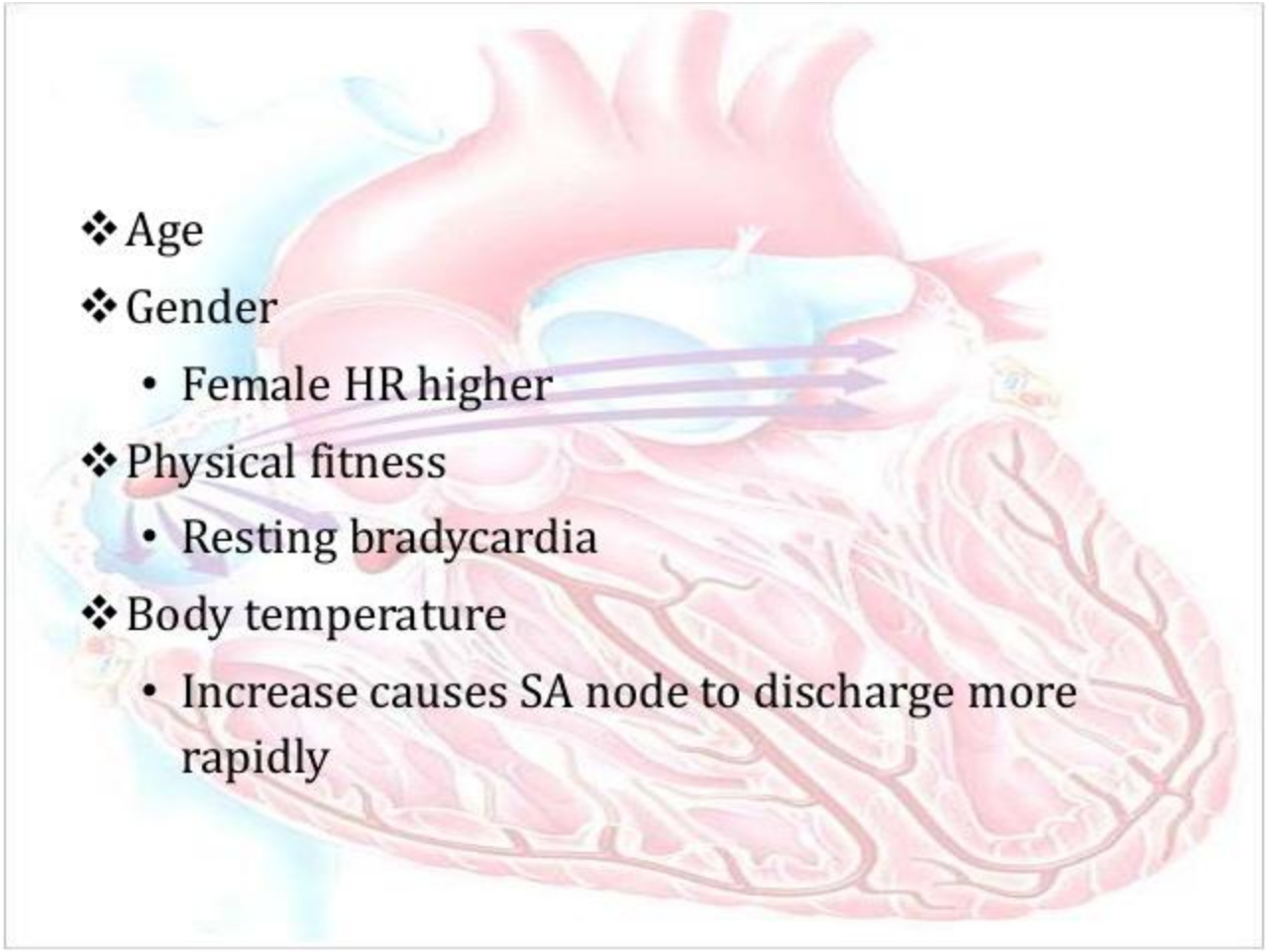
Posterior view

Blockade of coronary artery causes ***myocardial infarction***, or ***heart attack***.



Factors contribute to regulation of heart rate

- Chemical regulation
  - Cardiac activity depressed by
    - Hypoxia
    - Acidosis
    - Alkalosis
  - Hormones
    - Catecholamines and thyroid hormones increase HR and contractility
  - Cations
    - Alterations in balance of K^+, Na^+ and Ca^{2+} alter HR and contractility



- Age
- Gender
  - Female HR higher
- Physical fitness
  - Resting bradycardia
- Body temperature
  - Increase causes SA node to discharge more rapidly

Regulation of Stroke Volume

- Stroke volume is determined by sympathetic stimuli making the myocardial muscle fibers contract with greater strength and parasympathetic stimuli having the opposite effect.
- The factors regulate stroke volume
  - **Preload:** the degree of stretch of heart muscle before it contracts.
  - **Contractility:** the forcefulness of contraction of individual ventricular muscle fibers.
  - **Afterload:** the resistance to ejection of blood from the ventricle.

Preload

- Preload is defined as the force acting to stretch the ventricular fibers at end-diastole.
- An increase in myocardial muscle fiber length is associated with an increase in the force of contraction and thus increases the stroke volume and cardiac output.
- Higher the preload, the higher the stroke volume will be.

Contractility

- **Contractility** refers to the force generated by the contracting myocardium.
- Increased contractility results in increased stroke volume.
- Contractility is enhanced by circulating catecholamines, sympathetic neuronal activity, and certain medications
- Contractility is depressed by hypoxemia, acidosis, and certain medications (eg, beta-adrenergic blocking agents)

Afterload

- Afterload is the resistance to ejection of blood from the ventricle.
- The ejection of blood from the heart begins when pressure in the right ventricle exceeds the pressure in the pulmonary trunk (about 20 mmHg), and when the pressure in the left ventricle exceeds the pressure in the aorta (about 80 mmHg). At that point, the higher pressure in the ventricles causes blood to push the semilunar valves open. The pressure that must be overcome before a semilunar valve can open is termed the afterload.
- An increase in afterload causes stroke volume to decrease.

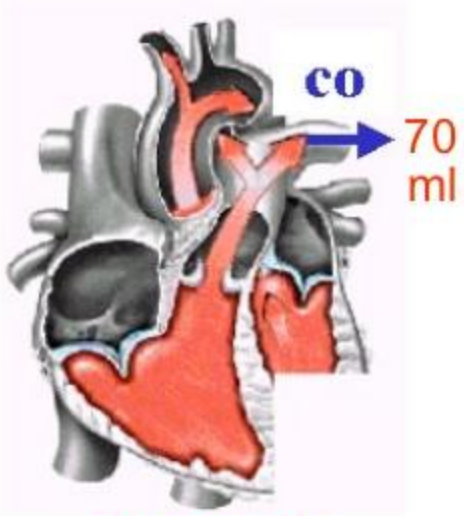
Cardiac Output (CO)

the amount of blood pumped out by each ventricle in 1 minute.

Cardiac output = stroke volume x heart rate

Example:

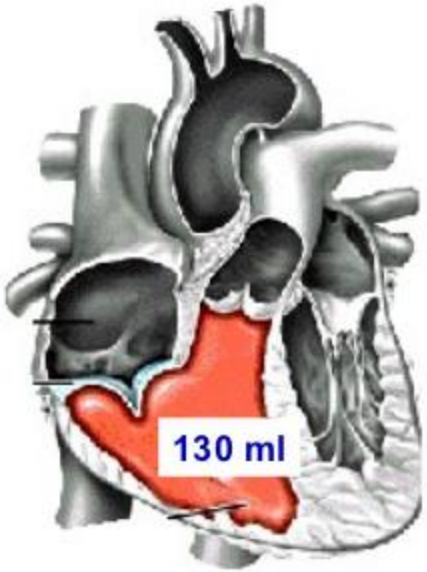
70 ml x 75 beat/min = 5,250 ml/min



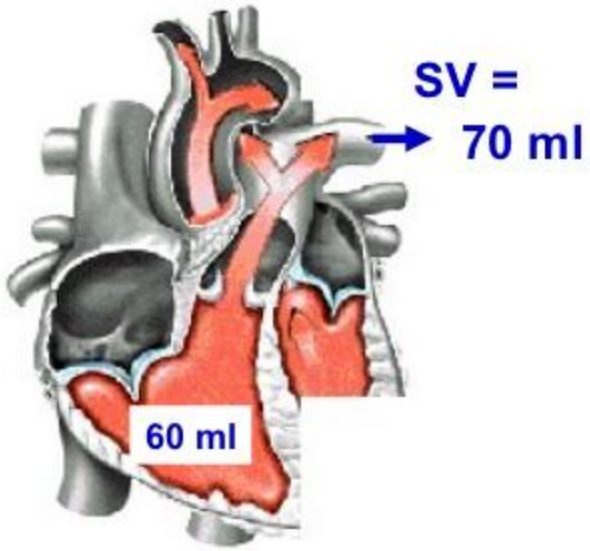
Ejection Fraction

= stroke volume ÷ end-diastolic ventricular volume

70 ml ÷ 130 ml = 54%



End of diastole

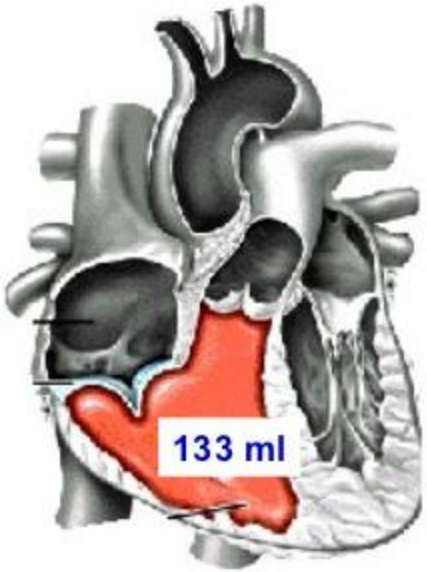


End of systole

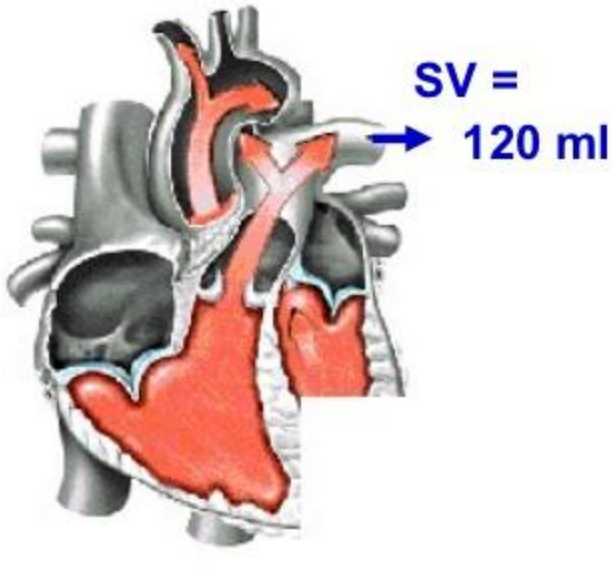
Ejection Fraction

increases during exercise

120 ml ÷ 133 ml = 90%



End of diastole



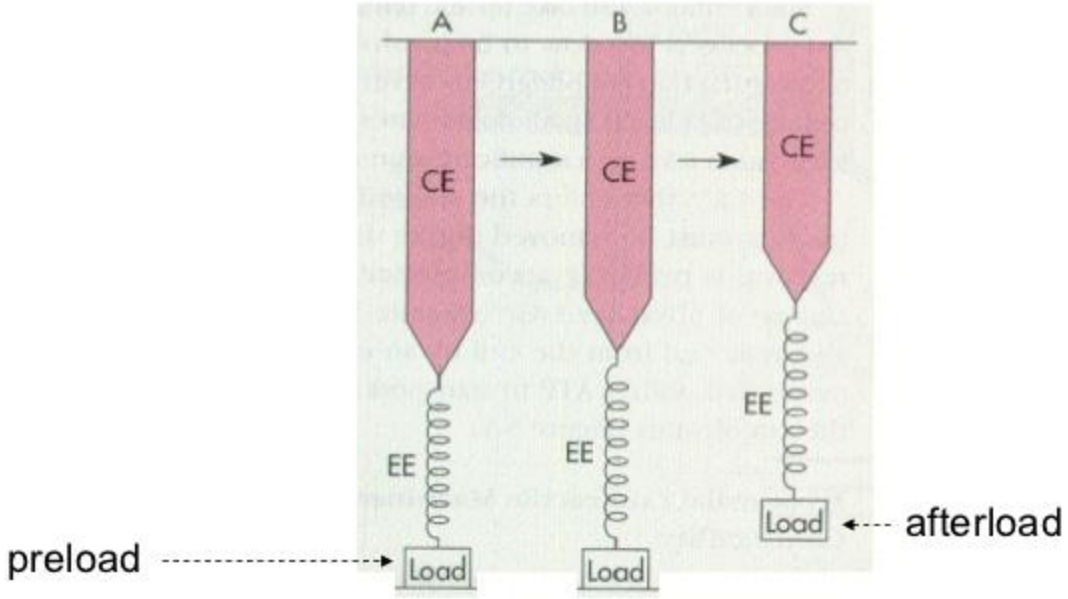
End of systole

Preload

the force that stretches the muscle before contraction.

Afterload

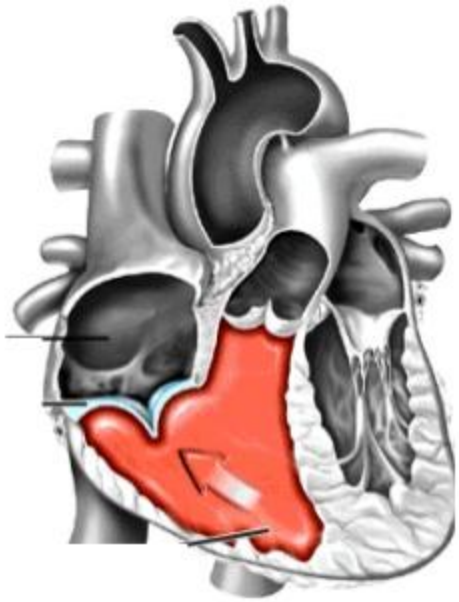
the force that stretches muscle during contraction.



Preload to ventricles = ***ventricular end diastolic pressure***

- the degree of stretch of the ventricular muscle cells just before they contract.

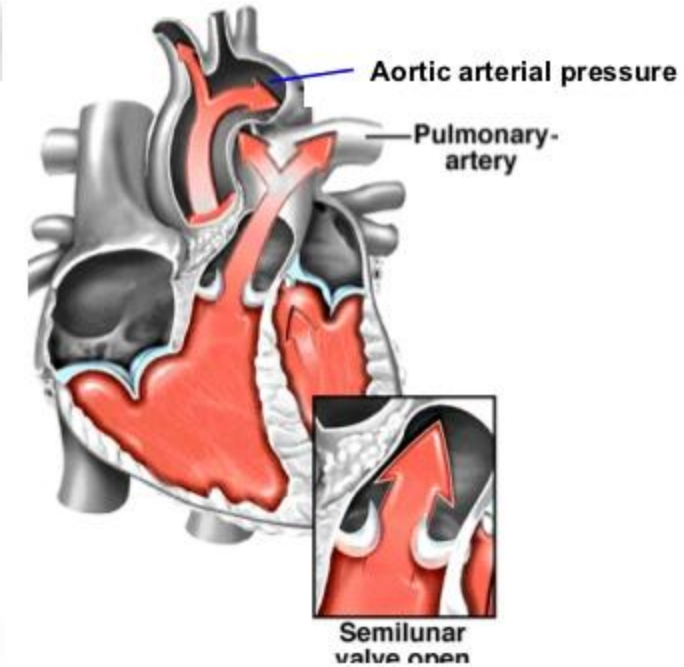
- determined by ventricular filling.



Afterload to left ventricle: ***aortic arterial pressure***

Afterload to right ventricle: ***pulmonary arterial pressure***

Afterload to the left ventricle is greater than that to the right ventricle.

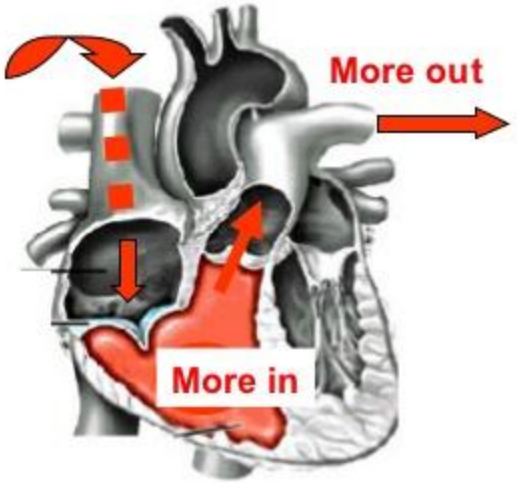


Factors on Cardiac Output

***5) Preload*:**

↑ Preload ⇒ ↑ cardiac output

(Starling-Frank Mechanism)

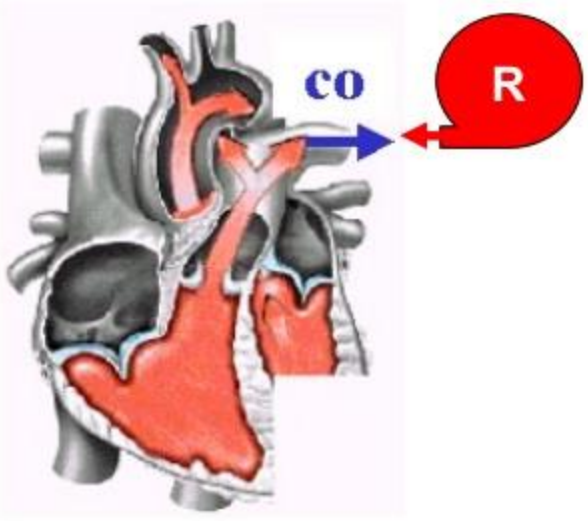


Factors on Cardiac Output

5) *Preload*:

2) ***Afterload***:

↑ afterload ⇒ ↓ CO



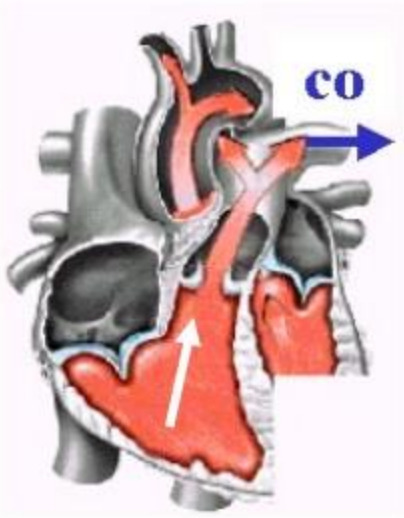
Factors on Cardiac Output

5) Preload:

2) *Afterload*:

3) ***Contractility***:

↑ contractility ⇒ ↑ CO



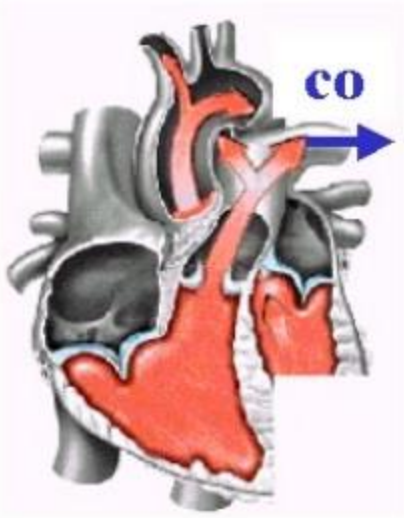
Factors on Cardiac Output

5) *Preload*:

2) *Afterload*:

3) *Contractility*:

4) ***Heart Rate***:



dual effects

↑CO = ↑Heart Rate x Stroke Volume

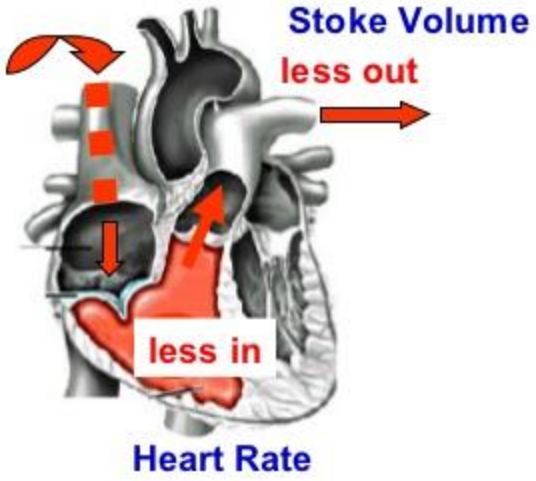
Factors on Cardiac Output

5) *Preload*:

2) *Afterload*:

3) *Contractility*:

4) ***Heart Rate***:



dual effects

↓CO = ↑Heart Rate x ↓Stroke Volume

300% 400%

Blood Pressure

- **Systolic:**

  - amount of pressure/force generated by LV to distribute blood into the aorta with each contraction of the heart

  - it is between 90 – 135 mmHg (120)

  - affected by CO and arterial distention.

- **Diastolic:**

  - amount of pressure/force sustained by the arteries during the relaxation phase of the heart

  - ability of the heart to rest while filling with blood

  - affected by peripheral vascular resistance

  - it is between 60 – 85 mmHg (80)

Blood Pressure

- Written as SBP /DBP
- Units mm Hg
- Mean Blood Pressure – {DBP + 1/3 PP}
- Pulse pressure – SBP - DBP

Classification of Hypertension

TABLE 1

Categories of Blood Pressure (BP) in Adults*

BP Category	Systolic Blood Pressure (SBP)		Diastolic Blood Pressure (DBP)
Normal	<120 mm Hg	and	<80 mm Hg
Elevated	120-129 mm Hg	and	<80 mm Hg
Hypertension			
Stage 1	130-139 mm Hg	or	80-89 mm Hg
Stage 2	≥140 mm Hg	or	≥90 mm Hg

* Individuals with SBP and DBP in two categories should be designated to the higher BP category. Blood pressure is based on an average of ≥2 careful readings obtained on ≥2 occasions.

Table 6, 2017 ACC/AHA Guideline on High Blood Pressure.

Cardiac auscultation
Heart Sounds

- Produced from blood turbulence caused by closing of the heart valves
- S1 – atrioventricular valve closure **LUB**
- S2 – semilunar valve closure **DUB**
- S3 – rapid ventricular filling
- S4 – atrial systole
- S3 is physiological less than 30 years of age.
- S4 is usually pathological.